Volume 25-02
15 January 1999

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OUTBREAK OF CRYPTOSPORIDIOSIS ASSOCIATED WITH A WATER SPRINKLER FOUNTAIN - MINNESOTA, 1997

Cryptosporidiosis associated with recreational water exposure is becoming recognized more freqently[1]. This report summarizes the investigation of a large outbreak of cryptosporidiosis associated with exposure to a water sprinkler fountain at the Minnesota Zoo. The initial cases were not diagnosed as cryptosporidiosis by the health-care system despite patients seeking care, underscoring the need for increased awareness of cryptosporidiosis and routine laboratory diagnostic practices among health-care providers.

On 10 July 1997, the Minnesota Department of Health (MDH) was notified by a parent about four cases of gastroenteritis among a group of 10 children whose only common exposure was a birthday party at the Minnesota Zoo on 29 June. The zoo provided MDH with a list of registered groups that had visited the zoo during 28 to 30 June; group members were contacted and interviewed about illness and zoo exposures. Initially, cases were defined as vomiting or diarrhea (defined as three or more loose stools during a 24-hour period) in persons who visited the zoo. Of 120 zoo visitors identified through the registered groups, 11 (9%) had illnesses that met the case definition. All had played in a water sprinkler fountain at the zoo, compared with seven (6%) of 109 controls (relative risk = undefined, $p < 0.001$). *Cryptosporidium* oocysts were identified in nine of 10 stool specimens of case-patients tested at MDH. Two of the laboratory-confirmed case-patients had submitted stool samples previously for ova and parasite examination to their health-care providers; both samples were reported as negative for parasites.

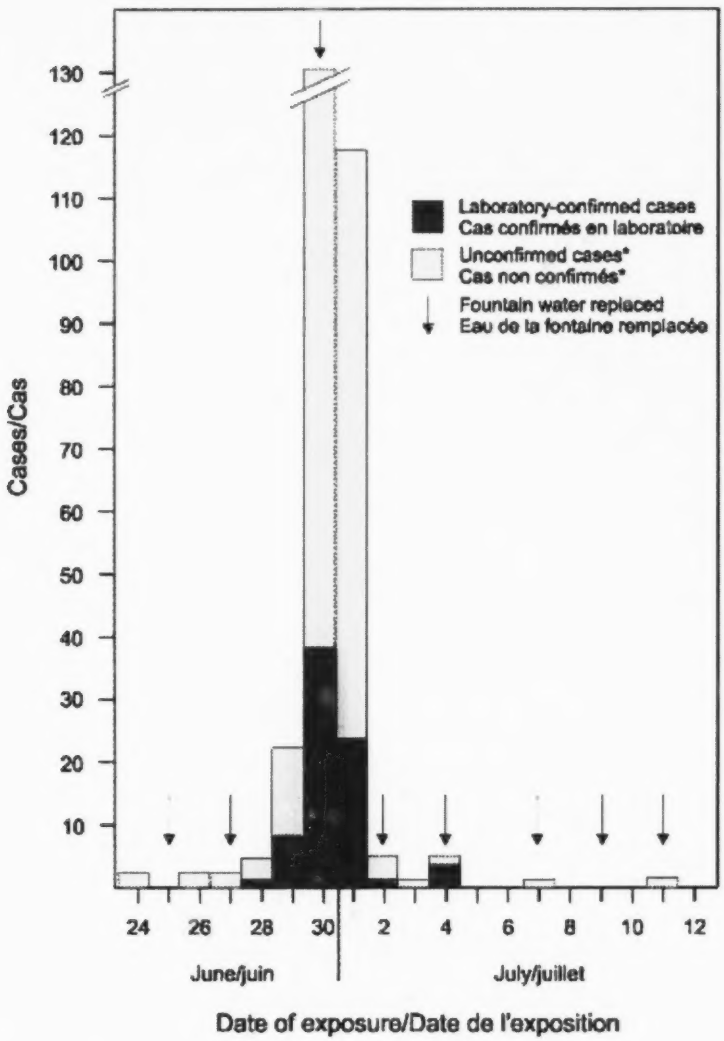
The fountain was closed on 11 July, and MDH issued a public statement advising persons who had visited the zoo and subsequently developed diarrheal illness to contact their physician and MDH. The public statement also stated that children who developed diarrhea after exposure to the fountain should not visit swimming beaches, swimming and wading pools, and other recreational water facilities until at least 2 weeks after recovery from diarrheal symptoms. MDH requested that all clinical laboratories in Minnesota specifically test all stools submitted for ova and parasite examination for *Cryptosporidium*, particularly during the outbreak.

A standard questionnaire was used to document illness history and zoo exposures in persons responding to the public statement. A revised case definition included persons with vomiting or diarrhea persisting at least 3 days, with onset 3 to 15 days after exposure to the zoo fountain. A total of 369 cases were identified, including the initial 11 cases; 73 (20%) were laboratory confirmed. Petting zoo exposure was reported by 191 (58%) of 332 case-patients, including 37 (55%) of 67 laboratory-confirmed cases. Age data were available for 351 case-patients; the median age was 6 years (range: 0 to 65 years), and 333 (95%) case-patients were aged <= 10 years. All but one of the 369 patients reported diarrhea; 317 (86%), abdominal cramps; 287 (78%), vomiting; 233 (63%), fever; and 11 (3%), bloody stools. The median duration of illness was 7 days. Six (2%) patients were hospitalized.

2007-02062/

Reported dates of fountain exposure for case-patients were from 24 June through 11 July (Figure 1). Exposure dates for confirmed case-patients were from 28 June through 1 July, with 68 (93%) exposures occurring from 29 June through 1 July (Figure 1). The median incubation period after fountain exposure was 6 days. In addition to case-patients with fountain exposure, nine laboratory-confirmed cases of cryptosporidiosis were identified among household contacts of case-patients with fountain exposure.

Figure 1 Reported cases of cryptosporidiosis associated with a water sprinkler fountain, by date of exposure - Minnesota, 1997+



+ n = 369.

* Defined as vomiting or three or more loose stools within a 24-hour period, with onset 3 to 15 days after fountain exposure and duration of at least 3 days

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The implicated water sprinkler fountain was designed and built as a decorative display in 1994. The fountain is comprised of 14 nozzles arranged in five rows and submerged beneath metal grates. The nozzles sprayed jets of water vertically approximately one to six feet. The water drained through the grates, collected in trenches, passed through a sand filter, was chlorinated, and then recirculated. The zoo routinely replaced the water every Monday, Wednesday, and Friday, but the filter was not flushed. Environmental health inspectors from MDH recommended the fountain not be used as an interactive play area. The zoo subsequently erected a fence around the fountain plaza and reopened it as a decorative display only. Water samples collected on 14 July were negative for *Cryptosporidium* oocysts.

The source of contamination of the fountain was not established, but contamination by a child wearing a diaper and playing in the fountain was suspected. Animals (including ruminants) in a petting zoo approximately 50 yards from the fountain tested negative for *Cryptosporidium* before being placed in the petting area and again during the outbreak investigation.

A 1997 survey of all clinical laboratories serving Minnesota residents indicated that 13 (22%) of 59 laboratories that perform ova and parasite examinations on site routinely test for *Cryptosporidium* as part of ova and parasite examinations (i.e. without a specific request from a physician). In a 1997 survey of physicians in Minnesota, 44 (79%) of 56 physicians who thought that their laboratory always tested for *Cryptosporidium* as part of an ova and parasite examination were incorrect.

MMWR Editorial Note

The findings in this report document a novel recreational water source for cryptosporidiosis. Outbreaks of cryptosporidiosis have been documented in a variety of other recreational water settings in the United States since 1988, including a lake, community and hotel pools, a large recreational water park, a wave pool, and a water slide[1]. As in several other outbreaks, there was no evidence in this outbreak that inadequate chlorination or filter malfunction contributed to transmission of *Cryptosporidium*. However, *Cryptosporidium* oocysts are resistant to disinfection by chlorine at levels generally used in recreational water, and recreational water filtration units that use sand filter media are not effective in removing the four- to six-micron oocysts[1]. The zoo fountain in this outbreak was designed as a decorative display and not an interactive play area. However, the fountain was a popular attraction for children on hot summer days. Children would commonly stand directly over the jets and soak their entire bodies, a practice which could explain contamination of the fountain and subsequent transmission associated with ingestion of water. Consumption of foods while walking in the fountain plaza was also a common practice.

Measures that might have reduced the risk for *Cryptosporidium* contamination of the fountain (e.g. showering before entering the fountain, excluding persons with diarrhea or incontinence, excluding children wearing diapers, and restricting food consumption in the fountain area) were not required or encouraged. Exclusion of persons from decorative water displays not designed for interactive use should be instituted and enforced. For recreational water facilities designed for human use, improved filtration may reduce risk.

Waterborne cryptosporidiosis is probably underrecognized and underreported [1]. Laboratory and physician surveys conducted in Minnesota indicate that most laboratories do not routinely test specifically for *Cryptosporidium* as part of ova and parasite examinations, even though many physicians assumed that they did. Even though cryptosporidiosis is reportable in Minnesota, this large

outbreak probably would have remained undetected if not for the parent reporting the cases to the health department. Two of the originally ill children had seen physicians, who ordered ova and parasite examinations; however, cryptosporidiosis remained undiagnosed until stool samples were examined specifically for *Cryptosporidium* at MDH. Because of their small size, *Cryptosporidium* oocysts can be difficult to detect by routine ova and parasite examination. The magnitude of this outbreak was probably determined only because of the public statement and the request that laboratories test all stools submitted for ova and parasite examination specifically for *Cryptosporidium*.

To better understand the magnitude of cryptosporidiosis, health-care providers should specfically request testing for suspected cryptosporidiosis. Laboratories should consider routinely testing for *Cryptosporidium* as part of their ova and parasite examination protocol. Alternatively, when reporting test results back to health-care providers, laboratories should specifically indicate when *Cryptosporidium* is not tested for as part of a requested ova and parasite examination. Cryptosporidiosis is reportable in 41 states; interpretation of national data would be facilitated by mandatory reporting in all states.

Reference

1. Anonymous. ***Cryptosporidium and water*: a public health handbook**. Atlanta, Georgia: Working Group on Waterborne Cryptosporidiosis, 1997.

Source: *Morbidity and Mortality Weekly Report, Vol 47, No 40, 1998.*

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Public Health Agency of Canada (PHAC)

Canada Communicable Disease Report CCDR

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Announcement

MEDICAL SERVICES BRANCH RESEARCH PROPOSALS

The Medical Services Branch is currently accepting research proposals contributing to the prevention and control of tuberculosis among Aboriginal peoples in Canada.

The selection process will be based on the degree of participation of Aboriginal people and of respect for culture, values, beliefs, and traditions. This request for proposals is open to all groups participating in research.

Candidates must submit a printed copy of their completed proposal. Proposals should include the primary investigator, and a description of the objectives, methods, budget, and timeline. They must be received by Health Canada no later than 1 March 1999. Research projects must begin 1 April 1999 and be completed by 31 March 2000.

Please send proposals to:

MSB Advisory Group for the Elimination of Tuberculosis
c/o Beth Kwavnick
Jeanne Mance Building
Postal locator 1920D, Tunney's Pasture
Ottawa ON
K1A 0L3

Our mission is to help the people of Canada maintain and improve their health.

Health Canada

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International Notes

HUMAN TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHIES

This article presents the conclusions and recommendations of the WHO Consultation on the global surveillance, diagnosis, and therapy of human transmissible spongiform encephalopathies which was held in February 1998 in Geneva.

Creutzfeldt-Jakob disease (CJD) is a rare and fatal human neurodegenerative condition characterized in most cases by a rapidly progressive dementia, myoclonus, and a periodic electroencephalogram (EEG). It is classified as a transmissible spongiform encephalopathy (TSE) because it causes characteristic spongy degeneration of the brain and can be transmitted to laboratory animals. TSEs also affect a range of animal species including sheep, goats, cows, deer, mink, and cats in non-experimental conditions. CJD is by far the most common human TSE. It occurs sporadically in about 85% of cases, and is inherited in 10% to 15% of cases; the remaining cases are iatrogenic. The other human prion diseases are Gerstmann-Sträussler-Scheinker disease and fatal familial insomnia, both extremely rare hereditary disorders, and kuru, a disease seen in Papua New Guinea and acquired via ritualistic cannibalism. CJD occurs worldwide, but as systematic surveillance has only been undertaken in very few countries, its incidence in much of the world is currently unknown.

Bovine spongiform encephalopathy (BSE), a TSE affecting cattle, was first reported in the United Kingdom in 1986, and over 170,000 cases have been reported since then in that country alone. Relatively small numbers of cases have also been reported in native-born cattle in Belgium, France, Ireland, Luxembourg, the Netherlands, Portugal, and Switzerland. Cases have also been reported in Canada, Denmark, the Falkland Islands, Germany, Italy, and Oman, but solely in animals imported from the United Kingdom.

In March 1996, the occurrence in the United Kingdom of 10 cases of an apparently new clinicopathologic variant of CJD (nvCJD) was announced. The temporal and geographic association with the BSE epidemic raised the possibility of a causal link. Evidence supporting this hypothesis has subsequently accumulated: (1) neuropathologic features similar to those of nvCJD are seen in macaque monkeys inoculated intracerebrally with brain material from confirmed cases of BSE; (2) transgenic mice (mice carrying only a human prion protein [PrP] gene) have now been shown to be susceptible to BSE; and (3) the biologic strain of the nvCJD agent (as defined by transmission characteristics of inbred strains of mice) and molecular 'strain' (as defined by the PrP glycosylation pattern) closely resemble the pattern in several animals which were naturally or experimentally infected with the BSE agent, but differ from those identified in sporadic CJD.

As the size of the human population exposed and suspectible to the BSE agent in the United Kingdom is not known, and there are uncertainties relating to the potential length and distribution of the incubation period, accurate prediction of the future number of nvCJD cases is not possible. Populations in other countries may also have been exposed to the infective agent as a result of importation of live cattle and/or cattle products or byproducts from BSE-affected countries. Thus, the possibility of a significant and perhaps geographically diverse nvCJD epidemic occurring over the next 2 decades cannot be dismissed at present.

At the time of the Consultation, a total of 24 cases of nvCJD had been reported to WHO, 23 in the United Kingdom and one case in France. Strong evidence indicates that nvCJD is linked with BSE and that the possibility of many more nvCJD cases

occurring in the future cannot be dismissed. Furthermore, and adding to the seriousness of the situation, no treatment is known to prevent the occurrence of CJD or halt disease progression. While there was concern at the possibility of a significant epidemic of nvCJD over the next 10 to 15 years, there was no lack of ideas for potential therapeutic interventions which had to be seriously evaluated. By considering them at the Consultation, WHO intended to stimulate the relevant bodies to further support research aimed at the early identification of an effective therapy. The conclusions and recommendations of the Consultation were as follows.

CJD clinical diagnosis: criteria for probable sporadic CJD

The clinical diagnosis of CJD is currently based upon the combination of progressive dementia, myoclonus, and multifocal neurologic dysfunction, associated with a characteristic period EEG. However in nvCJD, most growth hormone-related iatrogenic cases and up to 40% of sporadic cases do not have the characteristic EEG appearance. This hampers clinical diagnosis, and hence surveillance, and illustrates the need for additional diagnostic tests. Advances in CJD diagnostics have occurred in the past 2 years, in particular the assay for 14-3-3 protein in cerebrospinal fluid (CSF), which appears to have a high sensitivity and specificity for sporadic CJD diagnosis. The following criteria for probable sporadic CJD were proposed:

progressive dementia;

and

at least **two** out of the following four clinical features

- myoclonus
- visual or cerebellar disturbance
- pyramidal/extrapyramidal dysfunction
- akinetic mutism;

and

- a typical EEG during an illness of any duration;

and/or

- a positive 14-3-3 CSF assay and a clinical duration to death < 2 years;

and

- routine investigations should not suggest an alternative diagnosis.

Results from a recent study suggest that the detection of high signal from the basal ganglia on T2 and proton-density-weighted magnetic resonance imaging (MRI) supports the diagnosis of sporadic CJD. These abnormalities can be particularly prominent if a fluid attenuated inversion recovery sequence or diffusion-weighted images are obtained. The Consultation recommended that further research be conducted into the use of MRI in human TSE.

EEG interpretation

No widely agreed and validated definition of a diagnostic EEG tracing is available, leading to potential inconsistencies in case ascertainment between centres. To enhance CJD surveillance, a workable definition of a diagnostic EEG is required. It was proposed that the following criteria devised by Steinhoff and Knight be adopted now and results be further evaluated:

- strictly periodic activity
  - variations in intercomplex intervals are no higher than 500 ms
  - periodic activity is continuous for at least one 10-second period;
- bi- or triphasic morphology of periodic complexes;
- duration of majority of complexes 100 ms to 600 ms;
- periodic complexes may be generalized or lateralized but not regional or asynchronous.

New variant CJD: definition of a suspect case

New variant CJD cannot be diagnosed with certainty on clinical criteria alone at present. However, on the basis of the 23 neuropathologically confirmed cases, the diagnosis of nvCJD should be considered as a possibility in a patient with a progressive neuropsychiatric disorder with at least five out of the six clinical features given in Table 1. The suspicion of nvCJD is strengthened by the criteria given in Table 2. A patient with a progressive neuropsychiatric disorder and five out of the six clinical features in Table 1 and all of the criteria in Table 2 should be considered as a suspect case of nvCJD for surveillance purposes.

Pathologic diagnosis

The Consultation discouraged the use of cerebral biopsy in living patients except to make an alternative diagnosis of a treatable disease. It concurred with the previous WHO recommendation that instruments used for neurosurgery on patients with CJD should be destroyed. If reuse is unavoidable, instruments must be immersed in 1N NaOH or fresh undiluted hypochlorite for at least 1 hour, cleaned, and then autoclaved at 134° C for 1 hour.

A definite diagnosis of CJD, including nvCJD, is established only by neuropathologic examination. The Consultation recommended that autopsy be strongly encouraged in any suspect case of CJD. Where autopsy is not possible or permitted, postmortem biopsy of the brain should be sought.

Experience to date of the use of palatine tonsillar biopsy in CJD diagnosis is limited. Because the abnormal isoform of PrP has been detected in tonsillar tissue from patients with nvCJD but not patients with sporadic CJD, analysis of tonsillar tissue may provide potential diagnostic information in nvCJD, but requires further postmortem evaluation.

Notifiable Diseases Summary (Preliminary) - Sommaire des maladies à déclaration obligatoire (Provisoire)
New Cases Reported from 1 July - 30 September 1998 - Nouveaux cas déclarés du 1 juillet - 30 septembre 1998

Disease / Maladie	ICD-9 / CIM-9	Canada[7] J-S	Canada[7] Cum. 98	Canada[7] Cum. 97	Newfoundland / Terre-Neuve J-S	Newfoundland / Terre-Neuve Cum. 98	Newfoundland / Terre-Neuve Cum. 97	Prince Edward Island / Île-du-Prince-Édouard J-S	Prince Edward Island / Île-du-Prince-Édouard Cum. 98	Prince Edward Island / Île-du-Prince-Édouard Cum. 97	Nova Scotia / Nouvelle-Écosse J-S	Nova Scotia / Nouvelle-Écosse Cum. 98	Nova Scotia / Nouvelle-Écosse Cum. 97	New Brunswick / Nouveau-Brunswick J-S	New Brunswick / Nouveau-Brunswick Cum. 98	New Brunswick / Nouveau-Brunswick Cum. 97	Quebec / Québec J-S	Quebec / Québec Cum. 98	Quebec / Québec Cum. 97
AIDS-Sida	042-044			280	–	–	–			2			3	–	–	2			98
Amoebiasis - Amibiase	006	307	942	1387	–	–	5	1	2	1	3	19	14	–	–	5	45	135	179
Botulism - Botulisme	005.1	1	3	5	–	–	–	–	–	–	–	–	–	–	–	–	–	1	–
Brucellosis - Brucellose	023	3	7	10	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Campylobacteriosis - Campylobactériose	008.41	4189	9121	9677	98	174	77	21	38	33	80	175	173	93	223	175	890	1980	2219
Chancroid - Chancre mou	099.0																		
Chickenpox - Varicelle	052	1789	7045	22874	52	382	532	–	–	–	1	21	304	1	1	2	–	–	–
Chlamydia, genital - Chlamydiose génitale	099.81*	6384	20891	21958	102	282	251	41	112	105	307	915	829	258	700	587	1064	4137	4308
Cholera - Choléra	001	1	1		–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Diphtheria - Diphtérie	032			1															
Giardiasis - Giardiose	007.1	1098	3013	4090	12	38	31	3	5	5	30	68	89	17	58	102	185	550	844
Gonococcal infections - Infections gonococciques[1]	098	948	2942	3327	–	2	2	–	1	1	18	67	79	7	15	29	77	273	408
Gonococcal Ophthalmia neonatorum - Ophtalmie gonococcique du nouveau-né	098.4	8	18	45	–	–	–	–	–	–	–	–	–	–	–	–	1	2	1
Haemophilus influenzae B (all invasive) - (invasive) à H. influenzae B[20]	320.0,038.41*	8	35	40	–	–	–	–	–	–	–	–	1	–	–	–	–	10	8
Hepatitis A - Hépatite A	070.0,070.1	177	735	1391	1	2	3	–	1	–		8	12	–	3	2	22	133	395
Hepatitis B - Hépatite B	070.2,070.3	413	1201	1544	–	1	2			–	14	30	25	–	8	12	133	451	814
Hepatitis C - Hépatite C		3310	11815	13255	11	29	24	14	18	–	87	289	280	52	134	95	373	1785	831
Hepatitis non-A, non-B - Hépatite non-A, non-B		–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Legionellosis - Légionellose	482.41	24	52	58	–	–	–	1	1	–	1	3	1	2	2	–	8	12	12
Leprosy - Lèpre	030	1	3	4															
Listeriosis (all types) - Listériose (tous genres)	027.0,771.22*	14	31	32	1	1	–	–	–	1	–	–	–	1	1	–	–	–	–
Malaria - Paludisme	084	77	220	808	–	–	–	–	–	–	–	1		–	1	1	23	72	128
Measles - Rougeole	055	3	16	557	–	–	8	–	–	–	–	–	2	1	2	4	–	3	3
Meningitis, pneumococcal - Méningite à pneumocoques	320.1	13	31	28	–	–	1	–	–	2	–	–	1	–	–	–	–	–	–
Meningitis, other bacterial - Autres méningites bactériennes[5,6]		10	39	138	–	3	2	–	–	–	–	1	2	–	1	3	–	–	70
Meningitis/Encephalitis viral - Méningite/encéphalite virale[5]		250	333	188	–	–	–	–	1	1	–	3	1	2	2	–	4	18	109
Meningococcal infections - Infections à méningocoques	036	17	87	155	–	1	3	–	1	–	1	2	1	–	2	2	5	25	45
Mumps - Oreillons	072	18	78	240	–	–	–	–	–	–	1	2	2	–	1	2	2	8	18
Paratyphoid - Paratyphoïde	002.1-002.9	4	9	14														3	3
Pertussis - Coqueluche	033	1848	3201	2748	15	35	31	9	13	45	28	39	89	73	178	38	881	1345	504
Plague - Peste	020	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Poliomyelitis - Poliomyélite	045	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Rabies - Rage	071	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Rubella - Rubéole	056	3	81	3440	–	–	–	–	–	–	–	–	2	–	–	1	–	1	8
Congenital Rubella - Rubéole congénitale	771.0		1	1															
Salmonellosis - Salmonellose[18]	003	1806	4483	4429	38	148	34	10	29	27	57	148	84	43	115	98	211	779	848
Shigellosis - Shigellose	004	379	844	1108	–	–	2	–	–	7	1	7	7	4	11	9	38	198	319
Syphilis, Congenital - Syphilis congénitale	090	2	3		–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Syphilis, Early Latent - Syphilis, latente récente	092	–	3	8	–	–	–	–	–	–	–	–	–	–	–	–	–	1	2
Syphilis, Early Symptomatic - Syphilis, symptomatique récente	091	51	116	33	–	–	–	–	–	–	1	2	1	–	–	–	–	–	2
Other Syphilis - Autres syphilis	090,092-097	82	183	239	–	–	–	–	–	–	1	8	7	–	4	3	4	19	23
Tetanus - Tétanos	037	–	1	2	–	–	–	–	–	–	–	–	1	–	–	–	–	–	–
Trichinosis - Trichinose	124	13	19	1	–	–	–	–	–	–	–	3	2	–	–	–	31	155	187
Tuberculosis - Tuberculose	010-018	171	589	888	1	4	8	–	–	–	–	–	–	–	–	1	4	11	10
Typhoid - Typhoïde	002.0	11	37	38															
Verotoxigenic E. coli - E. coli vérotoxinogènes	008.01*	538	981	801	4	4	1	4	11	8	21	70	8	19	30	13	128	291	240
Yellow Fever - Fièvre jaune	060	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–

(1) Includes all 098 categories except 098.4.
(2) Includes buccal cellulitis or epiglottitis 464.3 in a child <5 years with no other causative organisms isolated.
(3) Includes encephalitis.
(4) All other categories except Haemophilus 320.2, Listeriosis 027.0, Meningococcal 036, Pneumococcal 320.1 and Tuberculosis 013.0.
(5) All categories except Measles 055, Mumps 072, Poliomyelitis 045, Rubella 056 and Yellow Fever 060
(6) Excludes Typhoid 002.0 and Paratyphoid 002.1 to 002.9.
* ICD-9 codes used in the list may be incomplete. All 5 digit codes are unofficial and are for LCDC surveillance purposes only.
† May not represent national total if data from the provinces are incomplete.

(1) Comprend toutes les rubriques 098, sauf 098.4.
(2) Comprend cellulite buccale ou épiglottite 464.3 chez un enfant < 5 ans chez qui aucun autre microorganisme causal n'a été isolé.
(3) Comprend encéphalite.
(4) Toutes les autres rubriques sauf à Haemophilus 320.2, listériose 027.0, à méningocoques 036, à pneumocoques 320.1 et tuberculeuse 013.0.
(5) Toutes les rubriques, sauf rougeole 055, oreillons 072, poliomyélite 045, rubéole 056 et fièvre jaune 060.
(6) Sauf typhoïde 002.0 et paratyphoïde 002.1 à 002.9.
* Les codes de la CIM-9 figurant dans la liste ne sont peut-être pas complets. Quant aux codes à 5 chiffres, ils ne sont pas officiels, ayant été établis uniquement aux fins de la surveillance du LLCM.
† Il se peut que ce chiffre ne représente pas le total national si les données provenant des provinces sont incomplètes.

New Cases Reported from 1 July - 30 September 1998 - Nouveaux cas déclarés du 1 juillet - 30 septembre 1998

Disease Maladie	ICD-9 CIM-9	Ontario			Manitoba			Saskatchewan			Alberta			British Columbia Colombie-Britannique			Yukon			Northwest Territories Territoires du Nord-ouest		
		J-S J-S	Cum. 98	Cum. 97	J-S J-S	Cum. 98	Cum. 97	J-S J-S	Cum. 98	Cum. 97	J-S J-S	Cum. 98	Cum. 97	J-S J-S	Cum. 98	Cum. 97	J-S J-S	Cum. 98	Cum. 97	J-S J-S	Cum. 98	Cum. 97
AIDS-Sida	042-044	_	_	101	_	_	_	_	_	2	_	_	34	_	_	17	_	_	_	_	_	_
Amoebiasis - Amibiase	006	140	430	729	11	39	32	18	38	34	8	33	58	84	241	309	1	2	3	_	3	_
Botulism - Botulisme	005.1	1	1	_	_	1	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	5
Brucellosis - Brucellose	023	2	5	1	_	_	_	_	_	_	_	_	4	_	_	_	_	_	_	1	2	5
Campylobacteriosis - Campylobactériose	008.41*	1819	3299	4044	88	179	175	105	219	177	454	881	584	932	1958	1998	4	8	9	7	11	13
Chancroid - Chancre mou	099.0	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_
Chickenpox - Varicelle	052	2093	6573	18757 7797	828	2181	1855	579	1754	1685	1513 750	8105 3284	4921 3733	_	_	_	32	88	128	190	470	232
Chlamydia, genital - Chlamydiose génitale	099.81*													_	_	_	58	134	129	308	819	717
Cholera - Choléra	001	1	1	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	1	_	_	_
Diphtheria - Diphtérie	032																					
Giardiasis - Giardiase	007.1	343	1026	1849	53	127	_	75	174	189	84	262	348	302	883	848	8	10	18	4	12	9
Gonococcal infections - Infections gonococciques[10]	098	343	1153	1439	107	325	318	91	255	224	84	302	398	183	431	339	2	5	_	58	113	102
Gonococcal Ophthalmia neonatorum - Ophtalmie gonococcique du nouveau-né	098.4	5	14	37	_	_	_	_	_	_	_	_	7	_	_	_	_	_	_	_	_	_
Haemophilus influenzae B (all invasive) - (invasive) à H. influenzae B[12]	320.0,038.41*	_	4	8	_	1	2	5	13	20	3	7	5	_	_	_	_	_	_	_	_	_
Hepatitis A - Hépatite A	070.0,070.1	54	179	353	8	29	51	8	29	182	7	58	182	79	288	229	_	1	2	_	5	_
Hepatitis B - Hépatite B	070.2,070.3	13	48	141	2	12	9	28	88	34	15	58	52	208	524	852	1	2	_	1	5	3
Hepatitis C - Hépatite C		819	3571	4894	_	_	_	170	554	483	434	1502	981	1235	3852	5581	10	60	48	5	23	18
Hepatitis non-A, non-B - Hépatite non-A, non-B		_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_
Legionellosis - Légionellose	482.41	14	28	33	_	_	1	_	_	_	_	5	9	_	_	_	_	_	_	_	1	_
Leprosy - Lèpre	030	_	1	3	1	1	_	_	_	_	_	1	1	_	_	_	_	_	_	_	_	_
Listeriosis (all types) - Listériose (tous genres)	027.0,771.22	8	23	29	_	_	_	3	5	2	1	1	_	_	_	_	_	_	_	_	_	_
Malaria - Paludisme	084	27	81	402	1	8	17	1	2	5	13	28	74	12	30	202	_	_	_	_	1	_
Measles - Rougeole	055	1	5	22	_	_	_	1	2	21	_	1	233	_	2	283	_	1	_	_	_	_
Meningitis, pneumococcal - Méningite à pneumocoques	320.1	_	_	_	2	5	2	1	4	1	3	9	8	3	8	10	_	_	1	4	5	_
Meningitis, other bacterial - Autres méningites bactériennes[13,14]		_	9	38	_	_	3	1	4	5	9	19	12	_	_	_	_	_	_	_	2	1
Meningitis/Encephalitis viral - Méningite/encéphalite virale[9]		_	1	11	99	111	20	43	58	5	91	123	29	10	19	11	_	_	_	1	1	1
Meningococcal infections - Infections à méningocoques	038	7	33	58	1	5	7	_	1	12	2	14	21	_	1	_	_	1	_	1	1	8
Mumps - Oreillons	072	5	24	53	_	_	2	3	14	8	4	18	28	3	12	129	_	1	1	_	_	_
Paratyphoid - Paratyphoïde	002.1-002.9	4	5	5	_	1	2						4									
Pertussis - Coqueluche	033	380	848	883	143	230	73	58	185	241	185	332	483	87	198	588	_	_	7	_	2	18
Plague - Peste	020	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_
Poliomyelitis - Poliomyélite	045	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_
Rabies - Rage	071																					
Rubella - Rubéole	056	1	13	25	1	20	3382	_	_	11	1	24	28	_	3	3	_	_	2	_	_	_
Congenital Rubella - Rubéole congénitale	771.0			1		1																
Salmonellosis - Salmonellose[15]	003	757	2024	2120	74	147	129	84	188	171	145	432	422	175	428	475	2	5	7	10	21	18
Shigellosis - Shigellose	004	92	253	307	88	169	85	48	85	72	84	110	71	88	110	228	_	1	2	_	2	1
Syphilis, Congenital - Syphilis, congénitale	090	_	_	_	_	_	_	_	_	_	2	3	_	_	_	_	_	_	_	_	_	_
Syphilis, Early Latent - Syphilis, latente récente	092	_	2	3	_	_	_	_	_	1	_	_	2	_	_	_	_	_	_	_	_	_
Syphilis, Early Symptomatic - Syphilis, symptomatique récente	091	2	3	9	_	_	_	_	_	_	1	2	5	47	109	18	_	_	_	_	_	_
Other Syphilis - Autres syphilis	090,092-097	51	112	182	_	_	_	_	_	_	8	21	44	_	_	_	_	1	_	_	_	_
Tetanus - Tétanos	037	_	1	1	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_
Trichinosis - Trichinose	124	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	13	19	1
Tuberculosis - Tuberculose	010-018	53	174	151	_	1	2	_	_	_	_	2	3	83	229	313	1	2	1	2	22	28
Typhoid - Typhoïde	002.0	7	21	22	_	1	2	_	_	_	_	2	3	_	2	_	_	_	_	_	_	_
Verotoxigenic E. coli - E. coli vérotoxinogènes	008.01*	125	251	328	35	87	53	21	32	32	118	149	114	85	88	_	_	_	_	_	_	8
Yellow Fever - Fièvre jaune	060	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_

SYMBOLS

. Not reportable
.. Not available
_ No cases reported

SIGNES

. À déclaration non obligatoire
.. Non disponible
_ Aucun cas déclarés

SOURCE:
Division of Disease Surveillance
Laboratory Centre for Disease Control
Health Canada
Ottawa, Ontario K1A 0L2
Tel.: (613) 957-0334

SOURCE:
Division de la surveillance des maladies transmissibles
Laboratoire de lutte contre la maladie
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Tél.: (613) 957-0334

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Table 1
Clinical features for definition of a suspect case of nvCJD

- Early psychiatric symptoms
- Early persistent paresthesia/dysesthesia
- Ataxia
- Chorea/dystonia or myoclonus
- Dementia
- Akinetic mutism

Genetic analysis

Screening cases of CJD for the mutations associated with the heriditary forms of disease raises ethic and logistic concerns. Written consent for genetic testing is considered mandatory in many countries but may be culturally unacceptable in others. The Consultation recommended that genetic counselling of patients and/or their families should be performed prior to any PrP gene analysis and that ideally written consent, or at least documented oral consent, should be obtained. The genetic counsellor should be provided with information on the genetics of the human TSE to be used when seeking consent. Because of the low PrP gene mutation detection rate in sporadic CJD, it is recommended that at present only those patients with a family history of a TSE be considered for PRP gene analysis as part of WHO's surveillance activities. Analysis could be performed at one of the proposed WHO collaborating centres (see below).

All suspect cases of nvCJD should undergo PrP gene analysis (if consent is obtained) to exclude a mutation and, for research purposes, to identify codon 129 status.

Geographic attribution of cases and proposed network of WHO collaborating centres

When a diagnosis of CJD is made, the initial geographic attribution should be the country of residence at the onset of clinical disease. Final attribution should be decided on a case-by-case basis.

As part of WHO's activities to promote the global surveillance of human TSE, the Consultation recommended that collaborating centres be established to aid in diagnosis and training.

Communication of information

The communication of any important new information to the public benefits from planning. This is particularly the case when the information is complex and has the potential to cause great concern. The Consultation recommended that each national authority plan a strategy for disseminating information that may result from CJD surveillance.

Therapy

The Consultation concluded that at present there is no available therapy known to alter the underlying disease process for any human TSE. Animal and in vitro studies have demonstrated that a number of therapeutic compounds have the potential for interfering with the underlying disease process. Although some compounds are known to delay the onset of disease (in some cases beyond the animal's natural life span), no compound is known that can cure a clinically affected animal.

Future evolution of new variant CJD

The Consultation noted that the possibility of a significant epidemic of nvCJD occurring within the next 10 to 15 years could not be dismissed and therefore emphasized that the early identification of an effective therapy was of paramount importance. Such a treatment would also offer hope to those individuals who are at risk of developing familial or iatrogenic disease.

Table 2
Criteria for definition of a suspect case of nvCJD

- Absence of a history of potential iatrogenic exposure
- Clinical duration > 6 months
- Age at onset < 50 years
- Absence of a PrP gene mutation
- EEG does not show the typical periodic appearance
- Routine investigations do not suggest an alternative diagnosis
- An MRI showing abnormal bilateral high signal from the pulvinar on axial T2- and/or proton density-weighted images

Further research

The Consultation stressed the pressing need for further research into the molecular properties of the TSE agent that could lead to potential disease-modifying compounds. In parallel, efforts should be made to identify presymptomatic diagnostic tests, to enable any future therapy to be used as early as possible in the disease course.

Source: *WHO Weekly Epidemiological Record, Vol 73, No 47, 1998*

SURVEILLANCE FOR CREUTZFELDT-JAKOB DISEASE IN CANADA

Health Canada conducts active surveillance for Creutzfeldt-Jakob disease (CJD) through the CJD Surveillance System (CJD-SS). Information collected from the surveillance system will be used to determine if there is any risk of developing CJD as a result of receiving a blood/blood product transfusion or following tissue transplantation. As a member of an international project team, CJD-SS Canada also conducts surveillance for new variant CJD (nvCJD). To date, 48 cases reported in 1997 and 1998 have been enrolled in the study. This includes some cases that have turned out not to be CJD.

Over the next several years, CJD-SS will continue to use active surveillance methods to seek out and investigate all cases of CJD occurring in Canada. Although we expect to be notified of cases primarily through neurologists, neuropathologists, and geriatricians, we ask that **any** physician aware of a case of CJD contact the surveillance system at our toll free number **1-888-489-2999**.

CJD occurs at a worldwide rate of between 0.5 and 1 case per million population per annum. There is an even distribution by sex, and the peak age of onset is between 60 and 65 years of age. Cases in persons < 30 years of age are rare. In Canada, the epidemiologic pattern and rate of CJD remains steady and consistent with the epidemiology in the rest of the world[1]. Current information on the epidemiology of CJD in Canada is derived from published Statistics Canada mortality data for the years 1979 to 1996 (CJD was not listed as a cause of death before 1979).

Overall, 421 deaths attributed to CJD were recorded in Canada in the 18-year period from 1979 to 1996, ranging from 14 to 34 per year, with a 1:1 male-to-female ratio. Eighty percent of deaths occurred in persons at least 60 years old, and 50% occurred in those 60 to 69 years of age, corresponding to the peak age of onset for sporadic-type CJD[1]. Thirteen deaths (3%), spanning the years 1979 to 1996, were reported in persons 30 to 44 years of age. No CJD deaths have been reported in persons < 30 years old in Canada.

New variant CJD has not been reported in Canada. As of the end of October 1998, 34 cases of nvCJD have been reported worldwide: 33 from the United Kingdom, and one case from France.

Reference

1. Will RG. *Prion disease surveillance*. In: Baker HF, Ridley RM, eds. *Methods in molecular medicine: prion diseases*. Totowa NJ: Humana Press, 1996:128.

Source: *E Stratton, MSc, Division of Blood-borne Pathogens, Bureau of Infectious Diseases, LCDC, Ottawa, ON.*

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